# Public Health: Chemical Exposure

# Specialist Report

# East Fork Boulder Creek Native Trout Restoration Project
# USDA-Forest Service-Dixie National Forest

Submitted by: /s/ Mike Golden

Mike Golden, Forest Fisheries Biologist
Dixie National Forest

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This report analyzes effects of the proposed East Fork Boulder Creek Native Trout Restoration project on public health relating to chemical exposure. The alternatives that are analyzed, including actions that are not part of the Forest Service decision but connected to the project, are described in Appendix 1.

## Description of Affected Environment and Analysis Methods

The affected environment includes members of the public who may be exposed to chemicals applied during the proposed project. Exposure would be directly or indirectly through the water in streams that would be potentially affected by the proposed project: East Fork Boulder Creek, West Fork Boulder Creek, and Boulder Creek. Lakes and reservoirs affected by activities connected to the proposed project include King's Pasture Reservoir and the pond in King's Pasture. Additionally, irrigation water fed by these streams and the Garkane penstock may also be affected by the proposed project.

The project area is within an active cattle allotment (Boulder allotment). One pasture within the allotment (Between-the-Creeks) contains flowing portions of streams within the project treatment area. A second pasture, Nazer Draw, contains portions of streams that may be affected by water flowing from the proposed treatment area. In addition to livestock management, people also use the project area for hunting, fishing, hiking, camping and other recreation.

Irrigation ditches leaving the area treated under the proposed project on the Boulder Irrigation Company's irrigation system were marked using a Garmin Rhino Global Position System (GPS) on May 27, 2010 with Loch Wade, the water master for Boulder Irrigation Company. In addition to the points of diversion, the points of entry to private property for actual use of the irrigation water were also marked using a Garmin Rhino GPS. With one exception, irrigation water is used for irrigating pastures for livestock consumption, as well as watering livestock in the first 5 miles (8 km) downstream from the penstock and Garkane Energy Boulder Creek Hydroelectric Project power plant (hydroplant) outflows. The exception is that some pasture vegetation on one private pasture is harvested for resale.

The municipal water supply of Boulder, Utah, comes from wells that are approximately 2 miles (3.2 km) straight line distance south of the hydroplant pond.

Boulder Creek passes by Boulder approximately 0.4 miles (0.6 km) east (straight line distance) of town and at this point is approximately 7 miles (11.3 km) downstream from the fish barrier on Boulder Creek at the lower end of the project area and 7.1 miles (11.4 km) downstream from the hydroplant pond outflow.

## *Direct and Indirect Effects*

### ***No Action and Non-chemical Treatment Alternatives***

Under the No Action and Non-chemical Treatment alternatives, no rotenone formulation or potassium permanganate neutralizer would be applied; therefore no direct or indirect effects to human health relating to chemical exposure would occur.

### ***Proposed Action***

Under the Proposed action, rotenone formulations and potassium permanganate would be applied as described in Appendix A and the specialist report for Chemicals and Application.

*Potential health effects of rotenone formulations*

In 2007 the U.S. Environmental Protection Agency (EPA) completed a re-registration eligibility decision for the use of rotenone and performed a risk-assessment for public health as part of that decision (USEPA 2006, USEPA 2007). Similarly, in 2008 the Forest Service completed an independently prepared risk-assessment for the use of rotenone and completed an analysis of public health effects as part of that assessment (Durkin 2008). In addition the potential human health risks associated with use of rotenone as a piscicide have been reviewed and assessed by several other authors in the past decade (Finlayson et al. 2000, Ling 2003, Entrix 2007, Fisher 2007, Turner et al. 2007, Ott 2008, Entrix 2010, Finlayson et al. 2010). Durkin (2008) was used as the primary reference for evaluating the human health risks of the proposed action. This assessment noted that in addition to toxicity studies that are relatively standard for pesticides, there is a large body of literature available on the neurotoxicity of rotenone with particular emphasis on the use of rotenone as an animal model for Parkinson's disease (Jenner 2001, Orr et al. 2002, Greenamyre et al. 2003, Hirsch et al. 2003, Perier et al. 2003, Trojanowski 2003, Uversky 2004, Höglinger et al. 2006, Gomez et al. 2007, Drechsel and Patel 2008). Additional literature supporting the contention that rotenone can have neurological effects has emerged since the Durkin (2008) assessment; therefore, these studies were also reviewed and incorporated into the potential impacts to human health from the Proposed action (e.g. Dhillon et al. 2008, Hancock et al. 2008, Cicchetti et al. 2009, Allen et al. 2010, Tanner et al. 2011).

As described in the Chemicals and Application Specialist Report, the liquid piscicidal formulations of rotenone that would be used under the Proposed Action contain inerts, adjuvants, metabolites, impurities, and contaminants in addition to the active ingredient rotenone. The known components of the potential liquid rotenone formulations to be used under the Proposed action are described in the Chemicals and Application Specialist Report and Durkin (2008). Durkin (2008) examined the potential negative effects of these compounds on humans. He concluded that metabolites, a breakdown

product of rotenone, did not increase the risk of human health effects associated with the use of rotenone formulations. Similarly, he concluded that available data indicate that the inerts are not present in amounts that would increase the risks associated with the proposed formulations. Durkin (2008) did find that adjuvants and impurities could increase that risk; however, because none of the formulations to be used under the Proposed action contain an adjuvant (e.g. piperonyl butoxide), there should be no impacts to human health associated with adjuvants under the Proposed action. The impact of impurities, such as degeulin and the "other associated resins," are considered in Durkin (2008).

Mechanism

Durkin (2008) characterizes the mechanism by which rotenone acts as being well described. Essentially, rotenone interferes with oxidative phosphorylation, a fundamental process in living cells in which nutrients are oxidized and the energy of oxidation is stored by the conversion of adenosine diphosphate (ADP) to adenosine triphosphate (ATP). While rotenone exposure will result in a decrease in ATP (i.e., an increase in ADP/ATP ratios), there is no indication that the toxicity of rotenone is based on bioenergetic deficits (Sherer et al. 2003; Uversky 2004). Rotenone inhibits a catalyst of the ADP to ATP conversion (NADH dehydrogenase) which resembles oxygen deprivation. This is not because of a direct blockage of oxygen uptake but because the blockage of NADH dehydrogenase prevents the use of oxygen in later stages of oxidative phosphorylation (Fontenot et al. 1994, Finlayson et al. 2000, Entrix 2007). The net result of rotenone poisoning at the cellular level is similar to oxygen deprivation and leads to anaerobic metabolism with the formation of lactic acid leading to acidosis. The central role of oxidative stress to the toxicity of rotenone is also supported by studies indicating that antioxidants can reduce or prevent expressions of rotenone toxicity (Inden et al. 2007; Nehru et al. 2008).

Acute toxicity

Data on acute oral toxicity of rotenone was reviewed in the both the EPA and Forest Service Assessments of rotenone (EPA 2006, EPA 2007, Durkin 2008). For characterizing the acute risks associated with oral exposures to mammalian wildlife, the EPA (2006) uses acute oral LD50 values of 102 mg/kg body weight in male rats and 39.5 mg/kg body weight in female rats. The lower LD50 value in female rats is associated with a lower excretion rate of rotenone. Other toxicity studies of rotenone formulations that yield somewhat lower LD50 values in terms of rotenone exposure — e.g., 6.5 rotenone mg/kg body weight in female rats — and in terms of combined rotenone and other extracts —e.g., 13 mg/kg body weight in female rats. In all studies, female rats appear to be somewhat more sensitive than male rats.

De Wilde et al. (1986) provide a relatively well-documented case report of a fatal accidental poisoning of a 3-year-old girl from a rotenone containing product, Galicide. Galicide's intended use was as an insecticide on animals. The girl ingested 10 ml of Galicide, which contains 6% rotenone. Assuming a bulk density of 1 g/mL as an

approximation, 10 mL of a 6% rotenone solution corresponds to 600 mg of rotenone. The body weight of the child is reported by De Wilde et al. (1986) as 15 kg; thus, they calculated a lethal dose of 40 mg rotenone/kg body weight (kg bw). This dose is virtually identical to the oral LD50 of 39.5 mg/kg bw of rotenone in female rats (EPA 2006). The correspondence between the rotenone oral LD50 for female rats and the lethal dose in a young girl may be coincidental, but the overall patterns in the acute lethal potency of rotenone do not suggest substantial species differences. Additionally, Wood et al. (2005) report on the fatality of a 47 year old female with extenuating health issues that died after consuming approximately 200 ml of another rotenone containing product, Bio Liquid Derris Plus (0.8% rotenone solution). If the rotenone poisoning was the sole cause of death, the estimated dose was 25 mg rotenone/kg bw.

Systemic and chronic toxicity

Durkin (2008) reviewed data on systemic and chronic toxicity and determined that the most significant study in terms of assessing human health affects was the chronic toxicity/oncogenicity study on which the EPA bases the chronic Reference Dose (RfD). In this study, rats were exposed to rotenone at dietary concentrations of 0, 7.5, 37.5, and 75 ppm for 2 years. The daily doses were estimated by the EPA at 0, 0.375, 1.88, and 3.75 mg/kg bw/day. The lowest dose, 0.375 mg/kg bw/day is classified as a "no observed adverse effect level" (NOAEL). Based on decreased body weight accompanied by decreased food consumption, EPA classifies the dose of 1.88 mg/kg bw/day as the "lowest observed adverse effect level" (LOAEL). Relative to a different route of exposure, Durkin (2008) indicated that rotenone is more likely to be toxic by inhalation than by oral exposure because inhalation exposures bypass initial metabolism and detoxification by the liver. Durkin (2008) cites studies submitted to the EPA in support of the registration of rotenone that report 4-hour LC50 values of 0.0235 mg/L in male rats and 0.0193 mg/L in female rats. As with the acute oral studies, female rats appear to be somewhat more sensitive than male rats to inhalation exposure to rotenone.

Durkin (2008) also reviews the potential for rotenone to affect the immune, endocrine, and reproductive systems, as well as its potential to be a mutagen and carcinogen. No studies were found suggesting that rotenone may have an effect on pathogen resistance with in vivo exposures. Weight loss is reported in several studies but appeared to be more related to toxicity than endocrine disruption; however, one study reported that intraperitoneal doses of 2 mg/kg bw/day to rats over a period of 30-60 days caused a decrease in plasma testosterone (Alam and Schmidt 2004). Although they attributed the effect to diminished bioenergetics, as well as general oxidative damage to adrenal and testicular tissue, not changes in thyroid or pituitary hormones, an alteration in testosterone levels would be regarded as a disruption in the endocrine system.

Durkin (2008) cites several studies showing potential for developmental impacts in rats and mice including: decreased body weight gain, increased unossified sternabrae, increased resorptions, and decreased fetal survival. The NOAEL for rats was identified

by EPA as 3 mg/kg bw/day, while the NOAEL for mice was identified at 15 mg/kg bw/day. Similarly, Durkin (2008) cites studies indicating the potential for reduced litter sizes and pup weights for rats, which produced an NOAEL of 2.4-3 mg/kg bw/day for litter size and 0.5-0.6 mg/kg bw/day for offspring weight.

Parkinson's disease and neurologic effects

Durkin (2008) acknowledges that there is a substantial body of literature concerning the use of rotenone to develop animal models for Parkinson's disease citing numerous published reviews (Jenner 2001, Orr et al. 2002, Perier et al. 2003, Trojanowski 2003, Greenamyre et al. 2003, Hirsch et al. 2003, Uversky 2004, Höglinger et al. 2006, Gomez et al. 2007, Drechsel and Patel 2008). Durkin (2008) points out that all of the early studies and many subsequent studies using rotenone to develop an animal model of Parkinson's disease involve routes of exposure that are not directly relevant to the human health risk from piscicide applications, such as the Proposed action (e.g. subcutaneous infusion, intravenous administration, or direct instillation into the brain).

Durkin (2008) focuses on a study by Inden et al. (2007) in which Parkinson's like effects were observed in mice after oral administration of rotenone by gavage (force-feeding through tube passed into stomach). They treated mice with gavage doses of 0, 0.25, 1.0, 2.5, 5.0, 10 or 30 mg/kg rotenone for 28 days. At doses of 10 and 30 mg/kg bw/day, effects included degeneration of dopaminergic neurons as well as decreased endurance in a rotorod test (a standard assay for motor function). Effects on dopamine neurons were sporadic at 10 mg/kg body weight but were seen in nearly all mice at 30 mg/kg body weight. Furthermore, Inden et al. (2007) discovered an accumulation of protein (synuclein) within viable neurons which may be consistent with Lewy body formation. Durkin (2008) recognized that the Inden et al. (2007) study showed adverse neurological effects, whether or not they are directly related to Parkinson's disease, may occur at oral doses of rotenone as low as 10 mg/kg bw/day (LOAEL) with an apparent NOAEL of 5 mg/kg bw/day.

While oral administration was able to reproduce some of the neurological effects seen with subcutaneous and intravenous administration in test animals, the same was not true for a study examining potential inhalation effects (Rojo et al. 2007). Rojo et al. (2007) inoculated mice intranasally with a 2.5 mg/kg dose of rotenone for 30 days. They found that rotenone did not produce any obvious motor alteration or damage to the nigrostriatal system.

Despite the publication of the Inden et al. (2007) study showing an oral dose of NOAEL below 10 mg/kg, Durkin (2008) used the most conservative acute and chronic reference doses of 0.015 mg/kg bw/day and 0.0004 mg/kg bw/day derived in the recent EPA re-registration eligibility document for the use of rotenone (USEPA 2007). One of the major reasons that these reference doses were adopted was EPA's use of an uncertainty factor of 1000 in their derivation. The uncertainty factor of 1000 was generated by multiplying together separate factors of 10 for each of three factors

considered as contributing to uncertainty: inter-species variability, intra-species variability, and uncertainties in the available data on rotenone. The factor for uncertainties in the available data reflects concern for the potential of rotenone to cause essentially permanent neurotoxic damage in pre-natal or early post-natal exposures, which might not induce observable adverse effects until late in life.

In addition Durkin (2008) discusses the scientific debate on the use of rotenone as an animal model for Parkinson's disease because of the broader spectrum of neurological effects induced by rotenone relative to the neurological effects seen in Parkinson's disease (Lapointe et al. 2004, Richter et al. 2007, Ravenstijn et al. 2008). The debate continues regarding if, and how, the rotenone animal model can be used to emulate the potential effects of Parkinson's Disease in humans (Cicchetti et al. 2009, Cicchetti et al. 2010, Greenamyre et al. 2010).

Since the publication of Durkin (2008), additional studies and reviews have been released supporting much of the earlier work showing that rotenone is a neurotoxin. Many involve routes of exposure not relevant to the human health risk under the proposed action (Allen et al. 2009, Drolet et al. 2009, Klintworth et al. 2009, Meurers et al. 2009, Mulcahy et al. 2011). The most germane studies are studies where the exposure route mimics those likely under the proposed action's application, as well as epidemiological studies of environmental risk factors elevating the risk of Parkinson's disease. Pan Montojo et al. (2010) offer information on relevant exposure routes not previously analyzed by EPA and Durkin (USEPA 2006, Durkin 2008). Pan Montojo et al. (2010) administered a rotenone solution to mice intragastrically with a stomach tube at a concentration of 5mg/kg bw 5 days a week for 1.5 to 3 months. They found that mice treated with rotenone produced alpha-synuclein accumulation in a number of nervous system structures. They also observed inflammation and alpha-synuclein phosphorylation in the enteric nervous system and the dorsal motor nucleus of the vagus. Finally, the mice treated with rotenone showed motor system impairment in a rotorod test.

Since the release of Durkin (2008), several epidemiological studies have been published postulating a link between rotenone exposure and Parkinson's like symptoms in humans (Hancock et al. 2008, Dhillon et al. 2008, Tanner et al. 2009, Tanner et al. 2011). Hancock et al (2008) conducted a case-control study of approximately 300 case and 300 control individuals that indicated an increased risk of Parkinson's disease with increasing pesticide exposure; however, they did not find a significantly increased risk for botanical pesticides, such as rotenone. Similarly, Dhillon et al.'s (2008) case-control study with 100 cases and 84 controls indicated an increased risk for Parkinson's disease for individuals that had used rotenone versus those that had not.

Prior to these studies, a consensus statement from a group of researchers regarding Parkinson's disease and the environment had determined that there was "limited suggestive evidence" that people exposed to pesticides had an increased risk of

Parkinson's disease and that there was "inadequate or insufficient evidence" to determine" whether people exposed to specific pesticides have an increased risk of Parkinson's disease (Bronstein et al. 2007). Essentially these researchers agreed that evidence suggested an association between pesticide exposure and increased risk of Parkinson's disease but that the body of evidence had bias and/or was confounding. They felt that the quantity, quality, and/or consistency of studies on specific pesticides were insufficient up to that time.

Similarly, Raffaele et al. (2011) discuss the benefits of and barriers to using epidemiological data in environmental risk assessments. They use studies of pesticide exposure contributing to the increased risk of Parkinson's disease as a specific example of barriers to using epidemiological studies for informing risk assessments. In particular they cite inconsistent findings between studies, generic categorization of pesticide exposure, and the use of dichotomous exposure categories (e.g. ever versus never) as reasons for difficulty in applying the findings of these studies to environmental risk assessments. They also note the difficulty in using epidemiological studies to evaluate a disease such as Parkinson's where multiple causal factors (genetic susceptibility, age, and environmental exposures) are present.

Many of the issues raised by Bronstein et al. (2007) and Raffaele et al. (2011) apply to the more recent epidemiological studies (Dhillon et al. 2008, Hancock et al. 2008, Tanner et al. 2009, Tanner et al. 2011). Conclusions differed among studies. For example, Dhillon et al. (2008) and Tanner et al. (2011) claimed a highly significant relationship between rotenone exposure and an elevated risk of Parkinson's disease, while Hancock et al. (2008) were unable to establish a significant relationship between botanical pesticide exposure and an elevated risk of Parkinson's disease and Tanner et al. (2009) did not find a relationship between rotenone use and elevated risk of Parkinson's disease. Study design was an issue in most of the studies, for example, Hancock et al. (2008) disclosed that their study design lacked statistical power because of the family-based case-control study design they employed. All four studies had relatively small sample sizes of individuals exposed to either botanical or organic pesticides including rotenone (anywhere from 2 to 51 people). Although they tried to control for it, Dhillon et al. (2008) identified several forms of bias involving self-reported data by subjects and case versus control knowledge of subjects by interviewers. Rugberg et al. (2011) noted a high potential for recall bias in their case-control study regarding pesticide use and its potential association with Parkinson's disease in Canada. They found that their "Cases" with Parkinson's disease overestimated, or over reported, their exposure to pesticides and were over 2.5 times more likely to identify pesticides as a cause of Parkinson's disease than the "Controls" without Parkinson's disease. Recall bias and misclassification bias may have influenced the results of some or all of these studies. Individuals with Parkinson's disease may be more likely to over report exposures and more likely to feel that exposure to a pesticide is directly related to Parkinson's disease. Past studies have attempting to establish links between pesticides and Parkinson's disease and the development a rotenone model to induce Parkinson's

like symptoms are numerous and widely available on the internet; therefore, cases with Parkinson's may be more likely to remember being exposed to pesticides or believe they have been exposed to pesticides or rotenone.

In the Dhillon et al. (2008) study there are also some concerns regarding the data reporting and design of questions and analysis regarding rotenone exposure. Of most concern is the question regarding the "use of organic pesticides such as rotenone in the past year." The authors were not clear whether test subjects just answered yes or no to that question or whether they specified that they were exposed to rotenone and not another "organic pesticide." The authors argue that elevated risk for Parkinson's disease may be attributable to rotenone based mostly on the responses to the question of "any rotenone use"; however, they also state that the question on "use of 'organic pesticides' such as rotenone in the past year" may reflect other pesticides that subjects considered as organic. It appears that the "any rotenone use" category was derived from combining subjects in the six categories related to rotenone/organic pesticides. Of these six questions the only one that showed a significantly increased risk was the question regarding the "use of organic pesticides such as rotenone in the past year." This particular question weighted the odds ratio for "any rotenone use" more than any of the other five questions, despite the fact that these cases and controls may not have been exposed to rotenone at all. If the "any rotenone use" category was derived from combining all subjects in the six categories of rotenone use, then it is difficult to explain why only three control subjects were reported for "any rotenone use" when 5 individual control subjects responded to a question regarding having "ever personally used, mixed or applied" a variety of products containing rotenone for "land use only." It would seem as though at least 5 control subjects should have responded to "any rotenone use" based on this, which would lower the odds ratio. Given the questions surrounding bias and study design, raised by authors themselves, these epidemiological studies do not provide a definitive causal link between rotenone exposure and an increased risk of Parkinson's disease. Additionally, relationship of the study to the Proposed Action is also of question, because the Dhillon et al. (2008) study had only 2 (maybe 3, although this is also unclear in the study) of the 100 case studies and none of the 84 control studies using rotenone in a fisheries context, and no information is provided on application methods or rates.

Of the epidemiological studies attempting associate rotenone use with Parkinson's disease, the recent study by Tanner et al. (2011) provides the best attempt at avoiding the potential pitfalls outlined by Bronstein et al. (2007) and Raffaele et al. (2011). They used data from a large study of agricultural pesticide applicators (Farming and Movement Evaluation [FAME] Study ) where some verification of self-reporting accuracy has been collected (Blair et al. 2002, Hoppin et al. 2002, Tanner et al. 2011). They determined "ever use" and "lifetime days of use" of a variety of different pesticides, including rotenone, by 110 subjects with medically verified Parkinson's disease (cases) and 358 subjects without diagnosed Parkinson's disease (controls). They found that 19 case subjects and 32 control subjects had been exposed to rotenone. The odds ratio

calculated in the study indicated that rotenone use was associated with a statistically significant elevated risk of contracting Parkinson's disease. They also found that having a first degree relative with Parkinson's disease was associated with a significantly elevated risk of contracting Parkinson's disease. This study had a larger sample size than many of the previous studies, but still only examined results for at total of 51 subjects exposed to rotenone. The study also may still contain recall bias, although some information does exist indicating that this bias may have been reduced with the larger FAME data set. The authors acknowledge that

Despite the limitations of the epidemiological study conducted by Tanner et al. (2011) it still adds to the suite of evidence suggesting that rotenone can have neurological effects. This evidence is acknowledged in rotenone risk assessments, such as Durkin (2008), as well as the recently published manual detailing Standard Operating Procedures for the use of rotenone (Finlayson et al. 2010). The Tanner et al. (2011) study is similar to much of the laboratory and epidemiological research in that it does not address the use of rotenone as a piscicide. In the case of Tanner et al. (2011) there entire subject group was primarily agricultural workers who often apply pesticides using different formulations, concentrations, and application techniques than would be used in a piscicide application such as the Proposed Action. As with the many risk assessments that have been conducted for the use of rotenone as a piscicide, Finlayson et al. (2010) concluded that while rotenone can indeed cause neurological problems, adverse effects are unlikely given the quick degradation time of rotenone, the small concentrations used during piscicide treatments and the limited exposure routes.

Effects on public health from potassium permanganate

Durkin (2008) considered the potential risks associated with using potassium permanganate to neutralize rotenone. Literature reviewed in the assessment indicated that potassium permanganate is a strong oxidizing agent, is irritating to the skin and respiratory tract, and can cause severe eye damage on direct contact. Literature also indicated excessive oral exposures to potassium permanganate can cause irritation to the gastrointestinal tract. Latent symptoms similar to Parkinson's disease were reported in a single case study; however, that case study used a concentration of potassium permanganate 230 to 455 times the concentration of potassium permanganate that would be added to detoxify rotenone. Additionally, as discussed in the Chemicals and Application Specialist Report, potassium permanganate will not persist in the water, because the oxidation reaction it has with rotenone will reduce it to potassium and manganese.

Effects on public health from post-oxidation elements

Durkin (2008) considered the potential human health effects of increased potassium and manganese concentrations in water following the oxidation reaction with rotenone. Both manganese and potassium are essential elements; however, excessive exposure to manganese can also cause neurological issues termed manganism or manganese-induced Parkinsonism. Durkin (2008) found that this neurotoxicity was well

documented for inhalation exposure but less so for ingestion exposure. The studies that were available suggested that in the absence of very high levels of background manganese levels, the small increase (generally 140-280 µg/l) in manganese associated using potassium permanganate as a neutralization agent should not elevate human health risks.

*Potential for public exposure from the Proposed action*

Public exposure routes to rotenone, the associated chemicals in rotenone formulations, and the potassium permanganate neutralizer as part of the proposed action consist of the following:

1) Dermal, inhalation, and possible ingestion exposure to non-pesticide applicators within the project area.
2) Dermal, inhalation, and possible ingestion exposure to non-pesticide applicators outside of the project area.
3) Ingestion exposure to non-pesticide applicators from consumption of fish, wildlife, livestock, and/or crops exposed to rotenone within, or downstream from, the project area.

Dermal, inhalation, and possible ingestion exposure to non-pesticide applicators within the project area

Durkin (2008) found that non-accidental acute exposure scenarios for the general public within a given project area where 200 µg/l active ingredient rotenone was being applied would modestly exceed the level of concern (hazard quotient central estimate = 1.3 upper bound = 1.9). The scenario that exceeded the level of concern involved a child drinking water from a treated water body. At the maximum active ingredient concentration (0.1 mg/l active ingredient) to be used under the Proposed Action, an 18.1 kg (40 pound) child would have to drink approximately 2.5 liters of water from the treated area during the treatment to reach the most conservative acute Reference Dose (0.015 mg/kg) offered by the EPA and accepted in the Forest Service Risk Assessment (USEPA 2007, Durkin 2008). The chronic reference dose is not germane since rotenone has not been shown to persist in flowing water and potassium permanganate will be used to neutralize the rotenone (Finlayson et al. 2001, Finlayson et al. 2010).

To reach the lowest observable effects level for neurological effects (5 mg/kg) seen in the Pan Montojo et al. (2010) study a 40 pound child would have to drink 913 liters (241 gal) of water from the treated area during the time of treatment. Finally to reach the lowest level (25 mg/kg) observed to cause mortality in a human (in association with other chemicals and health issues), a 40 pound child would have to drink 4,563 liters (1,205 gal) of water from the treatment area during the treatment. Therefore, it seems

unlikely that the general public would suffer ill effects unless a large amount of water was consumed directly from the treated area.

Such water consumption by the public should be highly unlikely under the proposed action. Public news releases and community postings prior to the treatment will inform the general public to avoid the treatment area during the treatment. Similarly, design criteria include EPA's recommendation of placarding to instruct the public not to enter the treatment area (EPA 2007). Rotenone exposure to the general public within the treatment area should be limited and consumption levels sufficient to cause ill effects unlikely; thus, the public health risk would be low.

Rotenone will leave the project area from three distinct places: Boulder Creek below the fish barriers, Garkane's penstock at the upper hydroplant, and the main hydroplant facility. The proposed action will use potassium permanganate to neutralize rotenone at all three of these locations. As discussed in the Chemicals and Application Specialist Report, application of potassium permanganate will oxidize rotenone and reduce potassium permanganate into less bioavailable compounds. As described in the Aquatic Biota Specialist Report, this reaction can often take 30 minutes to complete, so the project area must be extended for 0.25-0.5 miles (0.4-0.8 km) downstream from the neutralization stations.

Potassium permanganate ingestion can cause gastrointestinal irritation and possibly neurological damage but at considerably higher doses than those specified under the proposed action. Informing the public and restricting their access to the treatment area, per design criteria, should ameliorate the risk of the general public consuming water with potassium permanganate in it. Levels of elemental manganese and potassium will be temporarily increased in the treatment area but not to a level expected to increase human health risks.

<u>Dermal, inhalation, and possible ingestion exposure to non-pesticide applicators outside of the project area.</u>

Rotenone persistence in flowing waters has been shown to be relatively short, as sunlight and water turbulence caused by substrate, slope, and velocity all work to make rotenone persistence in running waters a function of travel time (Finlayson et al. 2001, Robertson and Smith-Vaniz 2008, Brown 2010). Combining the natural degradation rate of rotenone with the fact that the Proposed Action would use potassium permanganate to neutralize the rotenone formulation at all areas where water can exit the treatment area, makes exposure to the general public outside of the treatment area, and the 0.25 to 0.5 miles immediately downstream from the neutralization stations, limited to nonexistent. Contingency potassium permanganate stations will be used in the event that the main stations malfunction. Additionally, to ensure that neutralization is preceding properly, the sentinel fish monitoring procedures highlighted in Finlayson et al. (2010) will be used. The closest non-Forest lands receiving water

from the treated area are the hydroplant pond and the irrigation ditches exiting that pond. Informing the public and restricting their access to the treatment area, per design criteria, should ameliorate the risk of the general public consuming water with potassium permanganate in it. Forest and UDWR would work with Garkane to inform the public further and restrict access, if needed.

With the exception of the pasture fed by the upper hydroplant outflow, all private lands receiving water are over 0.5 miles downstream from neutralization sources and will not contain concentrations of rotenone or potassium permanganate high enough to be a public health concern (Finlayson et al. 2001, Durkin 2008, Finlayson et al. 2010). The private property pasture fed by the upper hydroplant outflow is approximately 0.3 miles (0.5 km) downstream from the proposed neutralization station. It is possible that the entire oxidation reaction may not be complete when water enters this property, meaning a small concentration of rotenone and or potassium permanganate may remain in the irrigation water on private property for a short distance. Sentinel fish will be deployed at the property line to determine whether concentrations of rotenone remain elevated. It would be expected that concentrations of rotenone and potassium permanganate would both be well below the level of concern by this point.

Levels of elemental manganese and potassium may be temporarily elevated downstream from the project area but not to a level that would increase human health risks (Durkin 2008).

As addressed in the Water Quality, Floodplains, and Wetlands Specialist Report, there are no drinking water surface protection zones or municipal watersheds directly within the East Fork of Boulder Creek and Boulder Creek from data received from the Utah Department of Environmental Quality Division of Drinking Water. Drinking water supplies would not be affected by the use of potassium permanganate because it rapidly breaks down into potassium, manganese, and water. In addition, no target streams are used directly as municipal or culinary water sources. As described in the Water Quality, Floodplains, and Wetlands Specialist Report, rotenone is strongly bound to organic matter, making it unlikely that it would enter ground water; therefore, it should not enter private or municipal well sources that supply drinking water to the local community.

<u>Ingestion exposure to non-pesticide applicators from consumption of fish, wildlife, livestock, and/or crops exposed to rotenone within or downstream from the project area</u>.

As highlighted above the use of the neutralization station will ameliorate any concerns with public consumption of crops watered downstream from the project area. The private land irrigation immediately downstream is for livestock pasture (Loch Wade, water master, Boulder Irrigation Co., personal communication 5/27/2010). Areas in Boulder Town that might use irrigation water to irrigate personal or commercial crops

for human consumption are over 5 miles (8 km) downstream from the closest neutralization station, which is well downstream of the area where active chemicals may still be present.

Durkin (2008) examined the potential bioconcentration of rotenone in fish exposed within a treatment area and found that the level of risk through human consumption of these fish was low. Any risk would be further ameliorated by informing the public and restricting public access during and shortly after the treatment. The public would be warned against consuming the fish, not just because of the rotenone but also because of hazards from bacterial growth in the dead fish. In addition to studies cited in Durkin (2008), Robertson and Smith-Vaniz (2008) also note that ill effects to humans through consumption of rotenone treated fish are highly unlikely.

It is possible that game animals that may be harvested for human consumption could consume water that has been treated with rotenone. In his review of rotenone toxicity, Ling (2003) found that rotenone is "not easily absorbed in higher animals and does not accumulate in the body." Absorption is relatively slow and, if absorbed, is broken down by the liver to less toxic excretable metabolites. Livestock that may be used for human consumption may also ingest water from the treated area; however, as discussed in the Range and Livestock Grazing Specialist Report, consumption by the livestock would be very low, because the overlap in period of use by livestock and presence of rotenone or potassium permanganate in the water would occur only until October 1, the latest date for use of the pasture with the treatment area, and in 2010 or 2012 but not in 2011 or 2013. Also, other water would be available for livestock in the area. Durkin (2008) reviewed literature regarding the absorption and excretion of ingested rotenone and it would suggest that bioaccumulation is not likely in exposed animals. With potential exposure of animals limited, potential for bioaccumulation in animals low, and human consumption of exposed animals low, human exposure to bioaccumulated chemicals in animals is even less likely. In addition at the concentrations to be used under the proposed action, an enormous volume of water from the treated area would have to be ingested during the 6-8 hour treatment time frame in order for a significant concentration of chemical to undergo uptake in any exposed animal.

## *Cumulative Effects*

### ***No Action and Non-chemical Treatment Alternative***

No direct or indirect effects will occur to public health under either the no action alternative or Non-chemical Treatment alternative; therefore there would be no cumulative effects from these alternatives.

***Proposed action***

Past, present, and reasonably foreseeable actions that would contribute to cumulative effects relating to chemical exposure from rotenone would be those actions in the CEA that involved use of the chemical. Rotenone has been used within the CEA several times in the last 10-15 years (Table 1). In each case a similar concentration of active ingredient rotenone and duration of treatment to that specified under the proposed action was used (Hepworth et al. 2000, Hepworth et al. 2001, Chamberlain and Ottenbacher 2008, Ottenbacher et al. 2009). Chronic exposure to rotenone has been shown to produce neurological effects; however, using exposure routes similar to what could be expected in the proposed project neurological effects have only been seen from repeated exposures over the course of 4-6 weeks. Each past treatment in the CEA occurred for one day in four of the last 11 years, resulting in potential acute exposure but not chronic exposure. Because there would be no chronic exposure from the proposed action, there would be no added effects and, thus, no cumulative effects to public health are expected.

**Table 1.** Water bodies treated with piscicidal formulations of rotenone within the CEA and the years they were treated.

| Water body | Treatment year |
|---|---|
| West Fork Boulder Creek | 2000, 2001 |
| Short Lake | 2007 |
| East Fork Boulder Creek | 2009 |

## *Summary*

Potential impacts to human health from exposure to rotenone have been recently reviewed by both the EPA during the re-registration process for rotenone use and by the Forest Service in relationship to the use of rotenone as a piscicide (USEPA 2006, USEPA 2007, Durkin 2008). Rotenone has been shown to have acute and chronic impacts to laboratory animals, and there are two documented cases of fatal rotenone poisoning in humans. Rotenone has been shown to be a neurotoxin in test animals when administered at certain amounts for certain time periods, in some cases producing symptoms similar to certain forms of Parkinsonism, although the routes of exposure and concentrations differ from those of the Proposed Action. Recent epidemiological studies claim to show a link between rotenone and Parkinsonism in humans; however, potential bias, study design, statistical power, and data interpretation issues confound the conclusions of these studies and their applicability to the proposed action (Dhillon et al. 2008, Tanner et al. 2011, Rugberg et al. 2011).

Furthermore, only 2 out of a total 184 case and control studies involved possible use of rotenone in a fish management context.

While rotenone and potassium permanganate have been shown to have potential impacts to human health the concentrations to be used, duration of application, and potential exposure routes in the proposed action limit the potential for human health impacts. Additionally, neutralizing rotenone with potassium permanganate, informing the public of treatment timing and location, and restricting public access to the treatment area would further ameliorate potential human health risks through reducing chemical exposure.

With no direct effects, including no chronic effects, there would be no cumulative effects from any of the alternatives. Although there is the potential for acute exposure to rotenone under the Proposed Action, under the application schedule, there would not be chronic exposure that could affect public health; therefore, there would be no cumulative effects under the Proposed Action.

## *Compliance with Other Laws and Regulations*

Compliance with laws and regulations affecting water quality are addressed in the specialist report on Floodplains/Wetlands and Water Quality. For the Proposed Action, design criteria 2, 3, and 4 ensure implementation meets all applicable regulations and policies.

## *Forest-plan Consistency Determination*

General direction #5 for Water Resource Improvement and Maintenance (F05 and 06) states "Limit use of herbicides, insecticides, rodenticides, or other chemicals which are harmful to . . . human health. Use these chemicals only when and where possible transport to surface water has a low probability of occurrence. Follow all label requirements concerning water quality protection." Under the Proposed Action, although the rotenone application will be made directly to surface water, harmful effects to human health would not be expected because of the limited to nonexistent exposure of the public to the chemicals. Following label requirements is required by the design criteria. The Proposed Action would be consistent with this general direction. The No Action Alternative and Non-Chemical Treatment Alternative do not involve the use of chemicals and, thus, are consistent with this general direction.

## *Use and/or consideration of Best Available Science*

This analysis considers the best available science. The analysis includes a summary of the credible scientific evidence which is relevant to evaluating reasonably foreseeable impacts. The analysis also identifies methods used and references scientific sources relied on.

The conclusions stated within this report are based on the scientific analysis that utilized a thorough review of relevant scientific information and a consideration of responsible opposing views. It is acknowledged that there may be incomplete or unavailable information, scientific uncertainty, and risk associated with the analysis included in this report.

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## Appendix 1. Project Area and Alternatives Analyzed in Detail

The following describes and compares the Forest Service alternatives analyzed. It includes a description of the UDWR's proposed project and considers UDWR's treatment alternative in detail. This section also presents the alternatives and the UDWR activities that would be authorized or connected actions to the alternatives in comparative form.

*Project Area*

The proposed East Fork Boulder Creek Native Trout Restoration Project (project) is located approximately 7 miles northwest of Boulder, Utah (see Figure 1). The total treatment area is as follows:

- approximately 7.8 miles (12.6 km) of East Fork Boulder Creek from the natural barrier (below headwater meadow) on East Fork Boulder Creek to its confluence with West Fork Boulder Creek;

- approximately 0.2 miles (0.4 km) of lower West Fork Boulder Creek, from a previously constructed barrier to its confluence with East Fork Boulder Creek;

- approximately 0.5 miles (0.8 km) of Boulder Creek from the confluence of East Fork Boulder Creek and West Fork Boulder Creek downstream to a previously constructed fish barrier;

- all seeps and springs flowing into those sections of streams proposed for fish removal; and

- the Garkane Energy water transfer pipeline between the West Fork Reservoir and King's Pasture Reservoir; King's Pasture (East Fork) Reservoir; a pond on private property in King's Pasture, and the Garkane Energy penstock, between King's Pasture Reservoir and the Garkane Energy Boulder Creek Hydroelectric Power Plant (main power plant).

The treatment stream reaches flow through portions of Sections 27, 28, 33, and 34 of T31S, R4E, and Sections 3, 10, 15, 21, 22, and 28 of T32S, R4E, Salt Lake Baseline Meridian. Treatment would include connecting waters, including relatively large inflows or tributaries with permanent fish habitat and smaller springs and seeps that are capable of at least temporarily holding small fish. Known tributaries and inflows vary in length from 10 meters to over 750 meters.

The reaches on NFS-lands are all on the Escalante Ranger District of the Forest in Garfield County, Utah. The inflow of the water transfer pipeline is at the West Fork Reservoir in Section 8, T32S, R4E, and the outflow is at King's Pasture Reservoir in Section 10 of T32S, R4E. The inflow of the penstock is at King's Pasture Reservoir, and the outflow is at the main power plant in Section 35 of T32S, R4E.

*No Action- No Further Treatment Scenario*

Under the No Action alternative, the Forest would not approve the pesticide use permit to UDWR, would not authorize UDWR to use motorized vehicles off of designated routes for the application of rotenone to waters of the treatment area on NFS lands, and would not approve a special use authorization for UDWR to bury removed fish.

The No Action alternative would not preclude UDWR from implementing actions on NFS lands that would meet the purpose and need for UDWR's project but do not require Forest Service authorization. This includes UDWR activities described under the Non-chemical Treatment alternative (Section 2.1.3) except for the use motorized vehicles off of designated routes or burial of removed fish on NFS lands. The No Action alternative would also not preclude UDWR from implementing actions on non-NFS lands that are related to the purpose and need for UDWR's project but not under Forest Service jurisdiction or authorization.

One possible option for UDWR is to take no further action to meet the purpose and need of the proposed project. This possible option is identified in this analysis as the "No Action - No Further Treatment Scenario" and is the basis for the effects analysis for the No Action alternative to provide the base line for comparison of expected future conditions if neither the Proposed Action nor Non-chemical Treatment alternative were implemented by the Forest and UDWR were to take no further action to meet the purpose and need.

*Proposed Action*

The Proposed Action is to approve the pesticide use permit that the Forest Service requires the UDWR to have to apply the fish toxicant rotenone to waters that flow on NFS lands and to authorize motorized vehicle use off of designated routes. The pesticide use permit would authorize the UDWR to implement a maximum of three treatments on NFS land, one treatment per year for three consecutive years. Waters on NFS land that would be treated by UDWR under the Forest Service pesticide use permit are as follows:

- approximately 7.8 miles (12.6 km) of East Fork Boulder Creek from the natural barrier (below headwater meadow) on East Fork Boulder Creek to its confluence with West Fork Boulder Creek;

- approximately 0.2 miles (0.4 km) of lower West Fork Boulder Creek, from a previously constructed barrier to its confluence with East Fork Boulder Creek;

- approximately 0.5 miles (0.8 km) of Boulder Creek from the confluence of East Fork Boulder Creek and West Fork Boulder Creek downstream to a previously constructed fish barrier; and

- all seeps and springs flowing into those sections of the stream reaches specified in the permit.

The UDWR activities that would be authorized by the Forest under the Proposed Action would completely eradicate non-native trout from East Fork Boulder Creek, a short segment of Boulder Creek, and a very short segment of West Fork Boulder Creek. All fish would be temporarily eliminated by UDWR from target waters. Use of motorized vehicles by UDWR off of designated routes may be needed to facilitate placement of equipment, especially neutralization equipment, in effective locations.

Several actions that are not part of the Forest Service decision are connected to the UDWR project, as follows. UDWR is proposing chemical treatment of connected waters on private property to meet the purpose of the UDWR project. Following fish removal, UDWR would introduce the CRCT into the treated stream segments to establish self-sustaining populations. Sterile hybrids of species of non-native trout may also be stocked by UDWR at some locations following the treatments to provide sport fishing

opportunities while native trout become established. The following describes the UDWR project in detail, including identification of those actions that do not require Forest Service authorization.

*Chemicals.* Liquid emulsifiable rotenone (Liquid Rotenone, 5% Active Ingredient, EPA Registration No. 432-172) would be used by UDWR to treat target waters. Rotenone was selected as the chemical to use because of its effectiveness in controlling fish populations and its lack of long-term effects on the environment (Sousa et al 1987). When used at the concentrations planned for the UDWR project, rotenone is a naturally occurring fish toxicant that is toxic to only fish, some aquatic invertebrates, and some juvenile amphibians. EPA found it to be not toxic to humans, other mammals, and birds at the concentrations used to remove fish (EPA 2007). It has been widely used in the United States since the 1950's. UDWR has used rotenone successfully in many similar projects and has refined application techniques to minimize adverse side effects to the environment (Hepworth et al. 2001a, Hepworth et al. 2001b, Hepworth et al. 2001c, Ottenbacher and Hepworth 2001, Chamberlain and Hepworth 2002a, Chamberlain and Hepworth 2002b, Chamberlain and Hepworth 2002c, Fridell et al. 2004, Fridell et al. 2005, Fridell and Rehm 2006).

Potassium permanganate would be used by UDWR to neutralize the rotenone at suitable locations to prevent the movement of rotenone into non-target waters. Potassium permanganate was selected, because it is a strong oxidizer that breaks down into potassium, manganese, and water. All are common in nature and have no deleterious environmental effects at the concentrations that would be used for the UDWR project activities, including those that would be authorized by the Forest under the Proposed Action (Finlayson et al. 2000). Potassium permanganate is used as an oxidizing agent in treatment plants to purify drinking water (EPA 1999). Although the oxidation process is not immediate, neutralization should occur within an estimated 0.25 to 0.5 miles of the neutralization site.

A more detailed description of the chemicals that would be used for the UDWR project activities, including those that would be authorized by the Forest under the Proposed Action, can be found in specialist report on Chemicals and Application of the Proposed Action.

*Application.* Liquid rotenone would be applied by UDWR at a rate of 0.5 to 2.0 ppm. In the pond and reservoir, liquid rotenone would be dispersed from personnel on small water-craft using pressurized backpack spray units. For flowing waters, seeps, and springs, liquid rotenone would be applied using a combination of 30 gallon and 5 gallon dispensers with constant flow drip-heads at approximately 50 to 60 stations throughout the UDWR project area over a 3 to 24 hour period (Finlayson et. al 2000, Ottenbacher et al. 2009). One 30 gallon drip station would be used by UDWR at each at the following:

- lower end of the headwater meadow at the upstream end of the UDWR project area,
- approximately halfway between the headwater meadow and King's Pasture Reservoir,
- immediately below King's Pasture Reservoir, and
- at the intake for the water flow pipeline between the West Fork Reservoir and King's Pasture Reservoir.

Five-gallon drip stations would be located by UDWR at approximately 1 mile intervals, beginning one mile below King's Pasture Reservoir and ending 1 mile upstream from the fish barriers on the main stem of East Fork Boulder Creek, and at all major springs and seeps within the UDWR project area. The

interval placement of drip stations on the main stem of East Fork Boulder Creek would be to facilitate efficient travel time of chemicals. Depending on flow volume, a single 30 gallon or 5 gallon drip would be placed by UDWR on the lower fish barrier on West Fork Boulder Creek. Pressurized backpack sprayers would be used by UDWR to apply a diluted solution of the chemical to springs and backwater areas containing fish that were not effectively treated by boat or drip station.

Rotenone would be neutralized by UDWR with potassium permanganate downstream from target waters. Three sites are planned: where the penstock water is released at the upper power plant, where water is released at the main power plant, and at the fish barrier at the lower end of the treatment area. Each site would have a main neutralization station and at least one contingency neutralization station to ensure effectiveness. The neutralization stations would prevent rotenone from escaping the target area, except for the estimated 0.25 to 0.5 miles downstream in which the neutralization or natural degradation of rotenone would be occurring.

*Post-treatment activity.* Following confirmation of complete non-native trout removal, UDWR would reintroduce CRCT into project stream reaches from "core" CRCT populations or from fish produced by UDWR CRCT brood stocks. Sterile hybrids of species of non-native trout may also be stocked by UDWR at some locations following the treatments to provide sport fishing opportunities while native trout become established. All UDWR transfers or stocking of fish would comply with Utah Department of Agriculture and Food rules and UDWR policies.

*Design Criteria.* The following design criteria would be implemented and included in the Forest Service authorizations:

1. Stream sections will be treated in the fall to minimize impacts on non-target wildlife species (amphibians, insectivorous birds and bats). The fall treatment period will also minimize the impacts on sport fishing recreation.

2. Each treatment will be preceded by internal and external notifications and media releases to notify the public of treatment sites and dates and will include the following: notification of the Boulder Town Council, notification of private landowners in the treatment area, and news releases in local papers.

3. The treatment area will be placarded to prohibit public access during treatment and for at least 3 days following treatment.

4. Application of the chemical will be conducted by licensed pesticide applicators in accordance with all applicable regulations and policies.

5. Access by motorized vehicles will be on National Forest System roads designated for motorized vehicle use to the extent possible. Any use of motorized vehicles off of designated routes will be minimal and will require written Forest Service approval.

6. Neutralization sites will be placed to maximize their effectiveness at preventing downstream escapement of rotenone.

7. Treated waters will remain open to fishing.

8. Transport to the site and storage of chemicals on the site will comply with FSH 2109.14.40 (Pesticide-Use Management and Coordination Handbook, Chapter 40 - Storage, Transportation, and Disposal).

9. Sentinel fish ("in situ bioassay") will be used for pesticide residues monitoring to determine the presence or absence of unacceptable environmental effects.

10. Treatments will be discontinued if the objective of complete removal of non-native trout from the project area has been met.

*Actions connected to but not included in the decision.* The following parts of the UDWR project, as described above, are not subject to Forest Service permit requirements, and therefore are not included in the Forest Service decision. Selection of the Proposed Action is for issuance of the pesticide use permit for the application of rotenone on NFS lands only. The following, however, are considered connected actions and thus included in the environmental analysis:

1. The proposed UDWR treatment area includes private property, including property owned by Garkane Energy; thus, this area is not under Forest Service jurisdiction. This includes approximately 1.4 miles of East Fork Boulder Creek, Kings Pasture Reservoir, and the pond in Kings Pasture. To meet the purpose and need of the UDWR project, these areas as well as the water in the transmission pipeline and penstock must be treated by UDWR. Forest Service approval of the pesticide use permit for UDWR to apply rotenone to waters on NFS land is not approval of UDWR activities on non-NFS lands; however, the Forest Service would not approve the pesticide use permit unless UDWR is able to complete its project by treating waters off of NFS land.

   The expectation is that the entire UDWR project treatment area would receive chemical treatment as described below, although the UDWR may decide to use another method or methods to achieve the treatment objective. FERC license order Section 4(e), item 16, condition 4, requires Garkane Energy to use its reasonable efforts to cooperate in the work of UDWR and other agencies to remove non-native fish and re-establish CRCT in the above stream sections. This cooperation has already been demonstrated through construction of the fish barriers and through the first chemical treatment of Kings Pasture Reservoir in 2009.

2. Stocking of fish is under the jurisdiction of UDWR; thus, the CRCT stocking is not under Forest Service jurisdiction. To meet the purpose and need of the UDWR project, the stream would need to be stocked by UDWR with CRCT from core populations or UDWR brood stock post-treatment.

   The expectation is that the post-treatment recolonization/stocking of CRCT would occur as described. The purpose and need for the UDWR project, including stocking with CRCT, is to implement conservation actions under the CRCT Conservation Agreement and Strategy, to which UDWR is a signatory. In addition, the Forest Service conditions regarding the non-native fish eradication and fish restocking were included in a 2006 settlement agreement relating to the FERC license conditions and signed by Garkane Energy, Forest Service, and UDWR.

3. Fishing regulations, including whether or not treated waters would remain open to fishing, is under the jurisdiction of UDWR.

The expectation is that UDWR would manage the fishing regulations to meet the conservation actions under the CRCT Conservation Agreement and Strategy. UDWR recognizes the importance of the area to recreation users. Because of this, UDWR may also stock sterile hybrids of species of non-native trout at some locations following the treatments while native trout become established.

*Non-chemical Treatment Alternative*

Under the Non-chemical Treatment alternative, the Forest Service would authorize UDWR to use motorized vehicles off of designated routes and approve a special use authorization for UDWR to bury fish that are removed as necessary to implement a non-chemical treatment to remove non-native trout from waters on NFS land.

The non-chemical treatment methods would not involve the use of rotenone or other pesticides on NFS lands and, therefore, would not require Forest Service approval. The effects of the non-chemical treatment are being analyzed, because this option may be exercised by UDWR in the event that the Forest Service were to choose not to authorize pesticide use, and the approach would be a connected action to the authorization of the use of motorized vehicles off of designated routes and approval of a special use authorization for burial of removed fish. The other connected actions that would also not require new Forest Service action are described below. UDWR's non-chemical treatment and other connected actions may or may not occur under the No Action alternative if the UDWR were to use motorized vehicles only on designated routes. These UDWR actions also may or may not occur under the Proposed Action.

Under the Non-chemical Treatment alternative, UDWR would use electrofishing to remove non-native trout from the treatment waters on NFS lands. Except for possible motorized vehicle use off of designated routes and burial of removed fish, this alternative would not require Forest Service authorization.

*Treatment area*. The treatment area would remain the same as described in the Proposed Action.

*Methodology and Equipment*. Electrofishing would be used by UDWR to remove non-native trout from the treatment area on NFS lands. Electrofishing introduces an electric current into the water and is commonly used as a fish removal method. The electricity causes an involuntary muscle contraction in the fish, attracting them toward the source of the electricity (electrode). Workers with long-handled nets then collect the stunned fish. Voltage, amperage, pulse frequency, and waveform are manipulated to maximize effectiveness, which can be influenced by water flow and velocity, temperature, clarity, conductivity (dissolved mineral content), and substrate. Other factors influencing effectiveness include the fish size, species and behavior, presence of aquatic vegetation, time of year, and time of day. It is most effective in shallow water and is, therefore, most commonly used in rivers and streams and occasionally in the shallow water zones of lakes.

Electrofishing removal would be accomplished by UDWR using multiple Smith-Root LR24 backpack electrofishing units or their equivalent from another manufacturer. Block nets of sufficient width would be set up to prevent fish emigration during removal activities. Dip nets, buckets, and live wells would also be necessary for capture and removal of brook trout (*Salvenlinus fontinalis*) and capture and safe holding of CRCT.

*Removal activities*. Mechanical removal of non-native trout species using backpack electrofishing has been attempted in several other projects (Moore et al. 1986, Meronek et al. 1996, Thompson and Rahel 1996, Buktenica et al. 2000, Kulp and Moore 2000, Shepard et al. 2002, Peterson et al. 2004, Moore et al. 2005, Meyer et al. 2006, Earle et al. 2007). The results of these prior mechanical removal projects indicate: 1) achieving complete mechanical removal of trout in streams with the width, complexity, and number of small, heavily vegetated springs/tributaries found in East Fork Boulder Creek would be difficult; 2) success would be enhanced by implementing multiple-pass depletion removal efforts 3 to 4 times within the same year, and 3) success would be enhanced by treatment over multiple years (minimum of 2). For this UDWR project, the multi-year removal effort would involve a minimum of 5 to 6 people conducting multiple-pass removal efforts for the majority of summer and early autumn (late June to September) over a period of several years. While such removal efforts would undoubtedly cause major reductions in brook trout density and biomass, they may or may not result in complete eradication. UDWR would begin CRCT reintroduction efforts only when no brook trout are found within the project area.

The electrofishing removal by UDWR would follow the population monitoring methods used by Utah State University's Institute for Natural Systems Engineering, Utah Water Research Lab (INSE) during their Garkane-funded fish population monitoring on the Boulder Creek system (Hardy et al. 2009a, Hardy et al. 2009b). Personnel would electrofish approximately 100-meter reaches in 8.5 miles of the mainstem of East Fork Boulder Creek, West Fork Boulder Creek, and Boulder Creek along with all spring inflows and tributary streams. A block net would be placed across the upstream and downstream end of each reach to increase capture efficiency by preventing emigration. Up to 4 passes, or until no fish were collected, would be completed through each reach. Each pass would involve all personnel walking in the stream channel and on the banks while applying constant electric current to the water from at least two backpack electrofishers. All organisms within the stream would be subjected to the electric field. All non-native brook trout would be removed from the system, killed and buried. Any CRCT collected would be held in buckets/live wells and returned to the stream after completion of the 4 pass removal.

*Effort.* One crew would consist of at least 2 personnel using backpack electrofishers, 2 netters retrieving stunned fish, and 1 person with a bucket receiving and disposing of fish. Electrofishing batteries would be recharged using small gasoline powered generators. Based on their previous monitoring efforts, INSE estimated that in a 40 hour work week, 9 sites that were each 100 m long could be completed by a 5 to 6 person crew using the four pass methodology (C. Williams, Institute for Natural Systems Engineering, personal communication with M. Golden, Dixie National Forest, 3/12/2010). Based on this INSE estimate, for UDWR fish removal activities under the Non-chemical Treatment alternative, one removal effort on the 11.5 km mainstem stream (12.8 reaches, 900 m long) on NFS land would require approximately 512 hours (12.8 reaches times 40 hours) or 63 days (8 hours per day) to be completed by a 5 to 6 person crew using the four pass method. An additional effort of approximately 13 days would be needed to treat the 2.3 km mainstem on private property.

Because UDWR's removal activities would need to occur between late-June or early July and September to minimize access, weather, and high stream flow issues, each removal effort would be limited to approximately 20 days to be able to conduct 4 removal efforts in a single year. To be able to treat the entire mainstem stream, on NFS lands and private lands, during any one removal effort, 20 people (four 5-person crews) would be needed. For four removal efforts, this would total up to 80 days per year. As described below, UDWR may need up to 10 years of removal effort under this method.

During the UDWR's 2009 chemical treatment of East Fork Boulder Creek above King's Pasture Reservoir, 23 relatively large inflows or tributaries with permanent fish habitat were identified, along with many smaller springs and seeps capable of at least temporarily holding small fish. These tributaries and inflows varied in length from 10 m to over 750 meters. Additional inflows and tributaries that contain fish habitat are probably present in the reach below Kings Pasture and could add another 30 days or more to the estimated treatment time.

Efficiency of fish removal by electrofishing is substantially lower in certain types of habitats found in the treatment area, especially those with heavy aquatic vegetation, root wads, woody debris, and boulder fields. The time for one removal effort in these types of areas could be higher, and effectiveness could be lower. Also, in order to eliminate the possibility of fish moving between treated and untreated reaches, crews would need to operate simultaneously, which may negatively impact fish-removal efficiency, as stream bed disturbance from upstream crews would impact water clarity and visibility for downstream crews. Because of reduced removal efficiency with electrofishing as the fish removal method, the UDWR project may extend to 10 years.

*Post-Fish Removal activities*. Post-fish-removal activities by UDWR would be the same as those described for the Proposed Action.

*Design Criteria.* The following design criteria would be included in the written authorization for use of motorized vehicles off of designated routes and the special use authorization for the burial of removed fish:

1. State of Utah decontamination protocols for prevention of the spread of Aquatic Nuisance Species will be followed for all gear and personnel involved with the removal project.

2. The Forest Archaeologist will be consulted about potential locations to bury fish to avoid impacts to cultural resources.

3. Dead fish collected will be buried no closer than 300 feet from the stream and away from known camping areas to minimize bear/human interactions.

4. Access by motorized vehicles will be on National Forest System roads designated for motorized vehicle use to the extent possible. Any use of motorized vehicles off of designated routes will be minimal, and will require written Forest Service approval.

5. Trails will be used whenever possible to move from one location to another to minimize soil and vegetation disturbance and to prevent establishing new trails.

6. Sensitive plant habitat will be avoided during action implementation.

7. Personnel will ensure reach being treated is void of livestock and people not involved with the operation. Treated waters will remain open to fishing.

*Actions connected to fish removal actions on NFS lands.* The following parts of the UDWR project, as discussed above, are not subject to Forest Service permit requirements, and therefore are not included in the Forest Service decision. They are considered connected actions to UDWR's fish removal activities on NFS lands and thus included in the environmental analysis:

1. As described for the Proposed Action, the UDWR treatment area includes private property, including that owned by Garkane Energy; thus, this area is not under Forest Service jurisdiction.

   The expectation is that under the Non-Chemical Treatment alternative, the UDWR would implement non-chemical treatment methods on non-NFS lands, as described below, although the UDWR may decide to use another method or methods to achieve the treatment objective on the private lands or not pursue treatment on the private lands. The flowing portions of the project area on private lands would undergo similar electrofishing removal by UDWR, as described for NFS lands above.

   For the non-flowing portions of the project area on private lands, electrofishing would not be effective in removing brook trout from King's Pasture Reservoir or the pond in Kings Pasture. To remove brook trout from these areas without use of chemicals, UDWR would deploy experimental gill nets with many different mesh sizes at several locations and depths throughout each water body. Other studies where this method has been successful at eradicating brook trout suggest that it would take at least two and up to four seasons of semi-continuous netting to eliminate all size classes of trout from small lakes with relatively low trout densities (Knapp and Matthews 1998, Parker et al. 2001).

2. Potential recolonization from East Fork Boulder Creek would severely reduce the efficacy of removing brook trout from King's Pasture Reservoir; therefore, UDWR would need to construct a fish migration barrier in East Fork Boulder Creek on private property above King's Pasture Reservoir.

   The barrier would generally consist of a small check dam constructed of boulders and large rocks, creating a vertical drop of approximately 5 ft on the downstream side. The location for the barrier would be selected by UDWR to utilize any naturally occurring drops which can be enhanced and where the stream channel and floodplain are confined to minimize the size of the structure and the amount of water impounded behind it. Barrier construction would comply with laws, regulations, and permitting requirements of the State Engineer for stream channel alteration. Barrier materials would be taken from the ground surface, near the stream. The collection of these materials would not require excavation, stream alteration, or vegetation disturbance. If sufficient material is not available on site, additional materials would be hauled to the barrier site from an approved source.

   The barrier location would be selected by UDWR to minimize changes in stream gradient, hydraulic function, and water pooling. In addition, the barrier would be constructed by UDWR adjacent to existing roads where equipment access is acceptable, thus requiring little disturbance to surrounding areas. Riparian vegetation would be disturbed as little as possible during the construction of the barrier, while areas where surface disturbance would occur would be restored to pre-project conditions. The barrier would not be placed in areas of cultural or historic significance or in areas where sensitive, threatened or endangered plants occur. It would be designed to operate under the natural fluctuations of a stream flow without routine maintenance. The barrier would be designed to pose little, if any, threat to the natural stream system or its associated riparian area so that if it were to fail, no damage would result to the stream environment. UDWR's maintenance could include

the adjustment or replacement of individual rock materials, but such work would be minor. The barrier could be removed but only after treatment is determined to be fully successful.

Neither netting nor electrofishing are options for UDWR for removing any non-native trout that may be using the upper portion of the penstock inflow or the lower portion of the pipeline from the West Fork Reservoir during treatment efforts. Shutting off water to these areas until they were completely dry would be the only way to ensure complete eradication; however, this is not feasible (M. Avant, Garkane Energy, personal communication with M. Golden, Dixie National Forest, 4/1/2010). Because of this, the effectiveness of the rest of the treatment would be reduced, contributing to the likelihood of the longer period of treatment.

3. Stocking of fish by UDWR would be as described for the Proposed Action.

4. As described for the Proposed Action, fishing regulations, including whether or not treated waters would remain open to fishing, is under the jurisdiction of UDWR. The expectation is as described for the Proposed Action.

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Figure 1. Project area location